	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error Definition	Errors
1	BRS	L1	54	pr-39	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/09 12:29			0
2	BRS	L2	6	pr-39 same (oligopeptide or analog)	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/09 12:30			0
3	BRS	L3	696	proteasome	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/09 12:45			0
4	BRS	L4	355	proteasome same inhibit$3	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/09 12:46			0
5	BRS	L5	2	1 same 4	USPAT; US-PGPUB; EPO; JPO; DERWENT	2002/11/09 12:46			0

=> d his

(FILE 'HOME' ENTERED AT 12:55:31 ON 09 NOV 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT
12:55:56 ON 09 NOV 2002
L1 392 S PR-39
L2 20 S L1 (P) (ANALOG OR OLIGOPEPTIDE)
L3 8 DUPLICATE REMOVE L2 (12 DUPLICATES REMOVED)
L4 24939 S PROTEASOME
L5 34 S L4 (P) ALPHA7
L6 11604 S L4 (P) INHIBIT?
L7 4 S L6 (P) ALPHA7
L8 10 S L1 (P) (L6 OR L7)
L9 6 DUPLICATE REMOVE L8 (4 DUPLICATES REMOVED)

=> log y

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FILE 'HOME' ENTERED AT 12:55:31 ON 09 NOV 2002

=> file medline caplus biosis embase scisearch agricola
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'MEDLINE' ENTERED AT 12:55:56 ON 09 NOV 2002

FILE 'CAPLUS' ENTERED AT 12:55:56 ON 09 NOV 2002
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FILE 'SCISEARCH' ENTERED AT 12:55:56 ON 09 NOV 2002
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FILE 'AGRICOLA' ENTERED AT 12:55:56 ON 09 NOV 2002

=> s pr-39
L1          392 PR-39

=> s l1 (p) (analog or oligopeptide)
L2           20 L1 (P) (ANALOG OR OLIGOPEPTIDE)

=> duplicate remove l2
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L2
L3             8 DUPLICATE REMOVE L2 (12 DUPLICATES REMOVED)

=> d l3 1-8 ibib abs

L3   ANSWER 1 OF 8  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         2001:489246  CAPLUS
DOCUMENT NUMBER:          135:87168
TITLE:                    Method for PR-39 peptide-mediated selective inhibition
                          of I.kappa.B.alpha. degradation
INVENTOR(S):              Simons, Michael; Gao, Youhe
PATENT ASSIGNEE(S):       Beth Israel Deaconess Medical Center, USA
SOURCE:                   PCT Int. Appl., 68 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
     WO 2001047540      A1   20010705       WO 2000-US35293  20001227
         W:  AU, CA, JP
         RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, TR
     EP 1242107         A1   20020925       EP 2000-989492   20001227
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, FI, CY, TR
PRIORITY APPLN. INFO.:                  US 1999-474967   A  19991229
                                        WO 2000-US35293  W  20001227
AB   The invention provides both a method and means for regulating
     I.kappa.B.alpha. degrdn., NF.kappa.B activity, and NF.kappa.B-dependent
     gene expression within living cells, tissues, and organs in-situ.  The
     selective regulation is performed using native PR-39 peptide or one of its
     shorter-length homologs, for interaction with such I.kappa.B.alpha. and
     proteasomes as are present in the cytoplasm of viable cells.  The result
```

of PR-39 peptide interaction with I.kappa.B.alpha. is a selective alteration in the intracellular proteolytic activity of proteasomes, which in turn, causes a redn. of I.kappa.B.alpha., a decrease of NF.kappa.B activity, and a down-regulation of NF.kappa.B-dependent gene expression.

L3 ANSWER 2 OF 8 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001663431 MEDLINE
DOCUMENT NUMBER: 21565666 PubMed ID: 11709430
TITLE: PR-39 and PR-11 peptides inhibit ischemia-reperfusion injury by blocking proteasome-mediated I kappa B alpha degradation.
AUTHOR: Bao J; Sato K; Li M; Gao Y; Abid R; Aird W; Simons M; Post M J
CORPORATE SOURCE: Angiogenesis Research Center, Dartmouth Medical School, Hanover, New Hampshire 03756, USA.
CONTRACT NUMBER: HL-53793 (NHLBI)
HL-636-09 (NHLBI)
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY. HEART AND CIRCULATORY PHYSIOLOGY, (2001 Dec) 281 (6) H2612-8.
Journal code: 100901228. ISSN: 0363-6135.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20011119
Last Updated on STN: 20020919
Entered Medline: 20020107
AB ***PR*** - ***39*** inhibits proteasome-mediated I kappa B alpha degradation and might protect against ischemia-reperfusion injury. We studied ***PR*** - ***39*** , its truncated form PR-11, and a mutant PR-11AAA, which lacks the ability to prevent I kappa B alpha degradation, in a rat heart ischemia-reperfusion model. After 30 min of ischemia and 24 h of reperfusion, cardiac function, infarct size, neutrophil infiltration, and myeloperoxidase activity were measured. Intramyocardial injection of 10 nmol/kg ***PR*** - ***39*** or PR-11 at the time of reperfusion reduced infarct size by 65% and 57%, respectively, which improved blood pressure, left ventricular systolic pressure, and relaxation and contractility (+/-dP/dt) compared with vehicle controls 24 h later. Neutrophil infiltration, myeloperoxidase activity, and the expression of intercellular adhesion molecule-1 and vascular cell adhesion molecule 1 were reduced. Thus ***PR*** - ***39*** and PR-11 effectively inhibit myocardial ischemia-reperfusion injury in the rat in vivo. This effect is mediated by inhibition of I kappa B alpha degradation and subsequent inhibition of nuclear factor-kappa B-dependent adhesion molecules. The active sequence is located in the first 11 amino acids, suggesting a potential for ***oligopeptide*** therapy as an adjunct to revascularization.

L3 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:706997 CAPLUS
DOCUMENT NUMBER: 133:276343
TITLE: Method for PR-39 peptide regulated stimulation of angiogenesis
INVENTOR(S): Simons, Michael; Gao, Youhe
PATENT ASSIGNEE(S): Beth Israel Deaconess Medical Center, USA
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000057895	A1	20001005	WO 2000-US7050	20000316
W: AU, CA, JP				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1165111	A1	20020102	EP 2000-919442	20000316
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1999-276868 A 19990326
WO 2000-US7050 W 20000326

AB The present invention provides both a method and means for regulating angiogenesis within living cells, tissues, and organs in-situ. The regulation is performed using native PR-39 peptide or one of its shorter-length homologs, for interaction with such proteasomes as one present in the cytoplasm of viable cells. The result of PR-39 peptide interaction with proteasomes is a decrease in the intracellular degrdn. of active peptides such as HIF-1.alpha. and a consequential stimulation of angiogenesis in-situ.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 8 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000020216 MEDLINE
DOCUMENT NUMBER: 20020216 PubMed ID: 10551808
TITLE: Lethal effects of apidaecin on Escherichia coli involve sequential molecular interactions with diverse targets.
AUTHOR: Castle M; Nazarian A; Yi S S; Tempst P
CORPORATE SOURCE: Molecular Biology Program, Memorial Sloan-Kettering Cancer Center, Cornell University, New York, New York 10021, USA.
CONTRACT NUMBER: P30 CA08748 (NCI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Nov 12) 274 (46) 32555-64.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000114
Last Updated on STN: 20000114
Entered Medline: 20000103

AB Apidaecins, short proline-arginine-rich peptides from insects, are highly bactericidal through a mechanism that includes stereoselective elements but is completely devoid of any pore-forming activity. The spectrum of antibacterial activity, always limited to Gram-negatives, is further dependent on a small number of variable residues and can be manipulated. We show here that mutations in the evolutionary conserved regions result in a more general loss of function, and we have used such ***analogs*** to probe molecular interactions in Escherichia coli. First, an assay was developed to measure selectively chiral association with cellular targets. By using this method, we find that apidaecin uptake is energy-driven and irreversible and yet can be partially competed by proline in a stereospecific fashion, results upholding a model of a permease/transporter-mediated mechanism. This putative transporter is not the end point of apidaecin action, for failure of certain peptide ***analogs*** to kill cells after entering indicates the existence of another downstream target. Tetracycline-induced loss of bactericidal activity and dose-dependent in vivo inhibition of translation by apidaecin point at components of the protein synthesis machinery as likely candidates. These findings provide new insights into the antibacterial mechanism of a unique group of peptides and perhaps, by extension, for distant mammalian relatives such as ***PR*** - ***39*** .

L3 ANSWER 5 OF 8 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 97273368 MEDLINE
DOCUMENT NUMBER: 97273368 PubMed ID: 9128101
TITLE: Synthesis and antibacterial action of cecropin and proline-arginine-rich peptides from pig intestine.
AUTHOR: Vunnam S; Juvvadi P; Merrifield R B
CORPORATE SOURCE: Rockefeller University, New York, New York, USA.
CONTRACT NUMBER: DK 01260 (NIDDK)
SOURCE: JOURNAL OF PEPTIDE RESEARCH, (1997 Jan) 49 (1) 59-66.
Journal code: 9707067. ISSN: 1397-002X.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 19970724
Last Updated on STN: 19970724

Entered Medline: 19970717

AB Two antimicrobial peptides, cecropin P1 (CP1), with a C-terminal carboxyl group, and ***PR*** - ***39*** , with an amidated, C-terminus, are found in the small intestine of the pig. Each is active against both Gram-positive and Gram-negative bacteria. We have synthesized these peptides and several ***analogs*** , including the D-enantiomers and the retro sequences, each with a free or acetylated amino terminus. The CP1 amide was also prepared. The retro CP1 peptides were much less active than the parent CP1 peptide, confirming the importance of sequence or the amide bond and helix dipole direction, and the N alpha-acetyl peptides were also less active, indicating that a free amino terminus is essential for high activity. The ratio of the lethal concentration of L/D isomers of CP1 is less than 1 for Gram-negative, but greater than 1 for Gram-positive bacteria. ***PR*** - ***39*** showed no significant chiral selectivity toward Escherichia coli, Bacillus subtilis and Streptococcus pyogenes, but the L/D ratio was high for Pseudomonas aeruginosa (66), and very high for Staphylococcus aureus (> 1000). In the latter case the lethal concentration for the D-isomer was 0.57 microM, whereas this organism was quite resistant to the L-isomer (> 600 microM). Thus the enantiomers of CP1 and ***PR*** - ***39*** are not equally active for all species. In a plate assay with a very small log-phase inoculum of Staph aureus, D- ***PR*** - ***39*** produced a clear zone of killing surrounded by a zone of stimulated growth. After prolonged incubation the two zones became one clear zone. Addition of D- ***PR*** - ***39*** to the wells of a dense turbid plate of growing cells showed a cleared zone for each of the test organisms, indicating that ***PR*** - ***39*** lyses the bacteria rather than simply inhibiting their multiplication.

L3 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:731865 CAPLUS
DOCUMENT NUMBER: 126:1218
TITLE: Synthetic peptides that inhibit leukocyte superoxide anion production and/or attract leukocytes
INVENTOR(S): Blecha, Frank; Shi, Jishu
PATENT ASSIGNEE(S): Kansas State University Research Foundation, USA
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9632129	A1	19961017	WO 1996-US4674	19960410
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN				
US 5830993	A	19981103	US 1995-419066	19950410
AU 9655345	A1	19961030	AU 1996-55345	19960410
PRIORITY APPLN. INFO.:			US 1995-419066	19950410
			WO 1996-US4674	19960410

AB Methods of inhibiting leukocyte O1- prodn. and attracting leukocytes using specific peptides are disclosed. These peptides include the proline-arginine (PR)-rich antimicrobial peptide known as ***PR*** - ***39*** and truncated ***analogs*** thereof. These peptides can be used as drugs that fight infection by attracting leukocytes to a wound site, yet restrict tissue damage at the wound site caused by excess oxygen radicals produced by these leukocytes.

L3 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:260860 CAPLUS
DOCUMENT NUMBER: 124:343935
TITLE: Chemistry of Pseudomonic Acid. Part 16. Aryl and heteroaryl ketone derivatives of monic acid
AUTHOR(S): Abson, Angela; Broom, Nigel J. P.; Coates, Philippa A.; Elder, John S.; Forrest, Andrew K.; Hannan, Peter C. T.; Hicks, Amanda J.; O'Hanlon, Peter J.; Masson,

Nicky D.; et al.
CORPORATE SOURCE: Smith[redacted]e Beecham Pharmaceuticals, Sur[redacted], RH3 7AJ, UK
SOURCE: Journal of Antibiotics (1996), 49(4), 390-4
CODEN: JANTAJ; ISSN: 0021-8820
PUBLISHER: Japan Antibiotics Research Association
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 124:343935
GI

/ Structure 1 in file .gra /

AB The synthesis, antibacterial activities, murine pharmacokinetic and infection model data for a range of aryl and heteroaryl ketone derivs. I [R = 2-, 3-, 4-R1C6H4, 5-R1-fur-2-yl, -3-yl, 6-R1-pyrid-2-yl, -3-yl, -4-yl, 2-R1-pyrimidin-5-yl, 5-R1-thien-2-yl, -3-yl, 2-R1-thiazol-5-yl, N-R1-2-oxothiazol-5-yl, R1 = H, OMe, OH, NMe2, SMe, S(O)Me, Ac, CF3, piperidino, pyrrolidino, Me, ***Pr*** (***39*** compds.)] of monic acid (I; R = OH) are reported. The best results were found for the 3-furyl and 2-methoxy thiazol-5-yl ***analogs*** .

L3 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1996:695862 CAPLUS
DOCUMENT NUMBER: 126:395
TITLE: Synthesis and study of the antimicrobial action of cecropin and proline-arginine-rich peptides from pig
AUTHOR(S): Vunnam, S.; Juvvadi, P.; Boman, H. G.; Merrifield, R. B.
CORPORATE SOURCE: Rockefeller University, New York, NY, 10021, USA
SOURCE: Peptides: Chemistry, Structure and Biology, Proceedings of the American Peptide Symposium, 14th, Columbus, Ohio, June 18-23, 1995 (1996), Meeting Date 1995, 233-234. Editor(s): Kaumaya, Pravin T. P.; Hodges, Robert S. Mayflower Scientific: Kingswinford, UK.
CODEN: 63NTAF
DOCUMENT TYPE: Conference
LANGUAGE: English
AB Two antimicrobial peptides, cecropin P1, with a C-terminal acid and ***PR*** - ***39*** with a C-terminal amide, were isolated from the small intestine of the pig and sequenced. Each is active against both Gram pos. and Gram neg. bacteria, with differences in their mechanism of action. To understand the importance of sequence, direction of amide bond, end group changes, chirality of the amino acids and handedness of the helix, we have synthesized these peptides and several ***analogs*** , including the D enantiomers and the retro sequenced, each with a free of acetylated amino terminus, and the CP1 amides.

=> d his

(FILE 'HOME' ENTERED AT 12:55:31 ON 09 NOV 2002)

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 12:55:56 ON 09 NOV 2002
L1 392 S PR-39
L2 20 S L1 (P) (ANALOG OR OLIGOPEPTIDE)
L3 8 DUPLICATE REMOVE L2 (12 DUPLICATES REMOVED)

=> s proteasome
L4 24939 PROTEASOME

=> s l4 (p) alpha7
L5 34 L4 (P) ALPHA7

=> s l4 (p) inhibit?
L6 11604 L4 (P) INHIBIT?

=> s l6 (p) alpha7

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L7            4 L6 (P) ALPHA7

=> s l1 (p) (l6 or l7)
L8           10 L1 (P) (L6 OR L7)

=> duplicate remove l8
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L8
L9             6 DUPLICATE REMOVE L8 (4 DUPLICATES REMOVED)

=> d l9 1-6 ibib abs

L9   ANSWER 1 OF 6  CAPLUS  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         2001:489246  CAPLUS
DOCUMENT NUMBER:          135:87168
TITLE:                    Method for PR-39 peptide-mediated selective inhibition
                          of I.kappa.B.alpha. degradation
INVENTOR(S):              Simons, Michael; Gao, Youhe
PATENT ASSIGNEE(S):       Beth Israel Deaconess Medical Center, USA
SOURCE:                   PCT Int. Appl., 68 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.         KIND  DATE           APPLICATION NO.   DATE
     ---------------    ----  --------       ---------------   --------
     WO 2001047540      A1    20010705       WO 2000-US35293   20001227
         W:  AU, CA, JP
         RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, TR
     EP 1242107         A1    20020925       EP 2000-989492    20001227
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, FI, CY, TR
PRIORITY APPLN. INFO.:                    US 1999-474967   A  19991229
                                          WO 2000-US35293  W  20001227
AB   The invention provides both a method and means for regulating
     I.kappa.B.alpha. degrdn., NF.kappa.B activity, and NF.kappa.B-dependent
     gene expression within living cells, tissues, and organs in-situ.  The
     selective regulation is performed using native PR-39 peptide or one of its
     shorter-length homologs, for interaction with such I.kappa.B.alpha. and
     proteasomes as are present in the cytoplasm of viable cells.  The result
     of PR-39 peptide interaction with I.kappa.B.alpha. is a selective
     alteration in the intracellular proteolytic activity of proteasomes, which
     in turn, causes a redn. of I.kappa.B.alpha., a decrease of NF.kappa.B
     activity, and a down-regulation of NF.kappa.B-dependent gene expression.

L9   ANSWER 2 OF 6     MEDLINE                              DUPLICATE 1
ACCESSION NUMBER:   2001663431     MEDLINE
DOCUMENT NUMBER:    21565666   PubMed ID: 11709430
TITLE:                ***PR***  -  ***39***   and PR-11 peptides
                      ***inhibit***   ischemia-reperfusion injury by blocking
                      ***proteasome***  -mediated I kappa B alpha degradation.
AUTHOR:             Bao J; Sato K; Li M; Gao Y; Abid R; Aird W; Simons M; Post
                    M J
CORPORATE SOURCE:   Angiogenesis Research Center, Dartmouth Medical School,
                    Hanover, New Hampshire 03756, USA.
CONTRACT NUMBER:    HL-53793 (NHLBI)
     HL-636-09 (NHLBI)
SOURCE:             AMERICAN JOURNAL OF PHYSIOLOGY. HEART AND CIRCULATORY
                    PHYSIOLOGY, (2001 Dec) 281 (6) H2612-8.
                    Journal code: 100901228. ISSN: 0363-6135.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
ENTRY MONTH:        200201
ENTRY DATE:         Entered STN: 20011119
                    Last Updated on STN: 20020919
                    Entered Medline: 20020107
```

AB ***PR*** - ***39*** ***inhibits*** ***proteasome*** -mediated I kappa B alpha degradation and might protect against ischemia-reperfusion injury. We studied ***PR*** - ***39*** , its truncated form PR-11, and a mutant PR-11AAA, which lacks the ability to prevent I kappa B alpha degradation, in a rat heart ischemia-reperfusion model. After 30 min of ischemia and 24 h of reperfusion, cardiac function, infarct size, neutrophil infiltration, and myeloperoxidase activity were measured. Intramyocardial injection of 10 nmol/kg ***PR*** - ***39*** or PR-11 at the time of reperfusion reduced infarct size by 65% and 57%, respectively, which improved blood pressure, left ventricular systolic pressure, and relaxation and contractility (+/-dP/dt) compared with vehicle controls 24 h later. Neutrophil infiltration, myeloperoxidase activity, and the expression of intercellular adhesion molecule-1 and vascular cell adhesion molecule 1 were reduced. Thus ***PR*** - ***39*** and PR-11 effectively ***inhibit*** myocardial ischemia-reperfusion injury in the rat in vivo. This effect is mediated by ***inhibition*** of I kappa B alpha degradation and subsequent ***inhibition*** of nuclear factor-kappa B-dependent adhesion molecules. The active sequence is located in the first 11 amino acids, suggesting a potential for oligopeptide therapy as an adjunct to revascularization.

L9 ANSWER 3 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2001:935656 SCISEARCH
THE GENUINE ARTICLE: 487UW
TITLE: ***PR*** - ***39*** and PR-11 peptides protect against ischemia-reperfusion injury by ***inhibition*** of ***proteasome*** mediated I kappa B alpha degradation
AUTHOR: Bao J L (Reprint); Gao Y H; Li M; Abid M R; Aird W; Simons M; Post M J
CORPORATE SOURCE: Harvard Univ, Beth Israel Deaconess Med Ctr, Sch Med, Boston, MA 02215 USA
COUNTRY OF AUTHOR: USA
SOURCE: CIRCULATION, (23 OCT 2001) Vol. 104, No. 17, Supp. [S], pp. 52-52. MA 251.
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA.
ISSN: 0009-7322.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0

L9 ANSWER 4 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:263290 BIOSIS
DOCUMENT NUMBER: PREV200200263290
TITLE: ***PR*** - ***39*** and PR-11 peptides protect against ischemia-reperfusion injury by ***inhibition*** of ***proteasome*** mediated IkappaBalpha degradation.
AUTHOR(S): Bao, Jialin (1); Gao, Youhe (1); Li, Min (1); Abid, Md. Ruhul (1); Aird, William (1); Simons, Michael (1); Post, Mark Johannes (1)
CORPORATE SOURCE: (1) Beth Israel Deaconess Med Ctr, Harvard Med Sch, Boston, MA USA
SOURCE: Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.52. http://circ.ahajournals.org/. print.
Meeting Info.: Scientific Sessions 2001 of the American Heart Association Anaheim, California, USA November 11-14, 2001
ISSN: 0009-7322.
DOCUMENT TYPE: Conference
LANGUAGE: English

L9 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:178321 CAPLUS
DOCUMENT NUMBER: 133:205925
TITLE: PR39, a peptide regulator of angiogenesis. [Erratum to document cited in CA132:149677]
AUTHOR(S): Li, Jian; Post, Mark; Volk, Rudiger; Gao, Youhe; Li, Min; Metals, Caroline; Sato, Kaori; Tsai, Jo; Aird, William; Rosenberg, Robert D.; Hampton, Thomas G.; Li, Jianyi; Sellke, Frank; Carmeliet, Peter; Simons, Michael

CORPORATE SOURCE: Angiogenesis Research Center, Department of Surgery, Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, 02215, USA
SOURCE: Nature Medicine (New York) (2000), 6(3), 356
CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature America
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The correct versions are given for Figs. 2a, c, and d on page 51; Fig. 3c on page 52; and Fig. 5b on page 53.

L9 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:46162 CAPLUS
DOCUMENT NUMBER: 132:149677
TITLE: PR39, a peptide regulator of angiogenesis
AUTHOR(S): Li, Jian; Post, Mark; Volk, Rudiger; Gao, Youhe; Li, Min; Metais, Caroline; Sato, Kaori; Tsai, Jo; Aird, William; Rosenberg, Robert D.; Hampton, Thomas G.; Li, Jianyi; Sellke, Frank; Carmeliet, Peter; Simons, Michael
CORPORATE SOURCE: Angiogenesis Research Center, Department of Surgery both at Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA, 02215, USA
SOURCE: Nature Medicine (New York) (2000), 6(1), 49-55
CODEN: NAMEFI; ISSN: 1078-8956
PUBLISHER: Nature America
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Although tissue injury and inflammation are considered essential for the induction of angiogenesis, the mol. controls of this cascade are mostly unknown. Here we show that a macrophage-derived peptide, PR39, inhibited the ubiquitin-proteasome-dependent degrdn. of hypoxia-inducible factor-1.alpha. protein, resulting in accelerated formation of vascular structures in vitro and increased myocardial vasculature in mice. For the latter, coronary flow studies demonstrated that PR39-induced angiogenesis resulted in the prodn. of functional blood vessels. These findings show that PR39 and related compds. can be used as potent inductors of angiogenesis, and that selective inhibition of hypoxia-inducible factor-1.alpha. degrdn. may underlie the mechanism of inflammation-induced angiogenesis.
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH, AGRICOLA' ENTERED AT 12:55:56 ON 09 NOV 2002
L1 392 S PR-39
L2 20 S L1 (P) (ANALOG OR OLIGOPEPTIDE)
L3 8 DUPLICATE REMOVE L2 (12 DUPLICATES REMOVED)
L4 24939 S PROTEASOME
L5 34 S L4 (P) ALPHA7
L6 11604 S L4 (P) INHIBIT?
L7 4 S L6 (P) ALPHA7
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